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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Jan 25	BLAST(R) searching in REGISTRY available in STN on the Web
NEWS 3	Jan 29	FSTA has been reloaded and moves to weekly updates
NEWS 4	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 5	Feb 19	Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 6	Mar 08	Gene Names now available in BIOSIS
NEWS 7	Mar 22	TOXLIT no longer available
NEWS 8	Mar 22	TRCTHERMO no longer available
NEWS 9	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS 10	Mar 28	LIPINSKI/CALC added for property searching in REGISTRY
NEWS 11	Apr 02	PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.
NEWS 12	Apr 08	"Ask CAS" for self-help around the clock
NEWS 13	Apr 09	BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS 14	Apr 09	ZDB will be removed from STN
NEWS 15	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 16	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 17	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 18	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 19	Jun 03	New e-mail delivery for search results now available
NEWS 20	Jun 10	MEDLINE Reload
NEWS 21	Jun 10	PCTFULL has been reloaded

NEWS EXPRESS	February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

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=> FIL BIOSIS MEDLINE SCISEARCH CA
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=> s (manganese superoxide dismutase or mnsod or catalase or cat or adcat or
phospholipid glutathione peroxidase or sod?)
L1     2103086 (MANGANESE SUPEROXIDE DISMUTASE OR MNSOD OR CATALASE OR CAT OR
               ADCAT OR PHOSPHOLIPID GLUTATHIONE PEROXIDASE OR SOD?)

=> s l1 and (antisense and inhibit?)
L2        1329 L1 AND (ANTISENSE AND INHIBIT?)

=> s l1 and (antisense (5n) inhibit?)
L3         438 L1 AND (ANTISENSE (5N) INHIBIT?)

=> s l1 and (antisense (3n) (inhibit? or reduc?)
UNMATCHED LEFT PARENTHESIS 'AND (ANTISENSE'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s l1 and (antisense (3n) (inhibit? or reduc?))
L4         425 L1 AND (ANTISENSE (3N) (INHIBIT? OR REDUC?))

=> s l1 and (antisense (3n) (inhibit? or reduc?)) and (antisense (5n) (start codon)
UNMATCHED LEFT PARENTHESIS 'AND (ANTISENSE'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s l1 and (antisense (3n) (inhibit? or reduc?)) and (antisense (5n) (start codon))
L5           0 L1 AND (ANTISENSE (3N) (INHIBIT? OR REDUC?)) AND (ANTISENSE
               (5N) (START CODON))

=> s l4 and (start codon)
L6           4 L4 AND (START CODON)

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7             2 DUP REM L6 (2 DUPLICATES REMOVED)

=> s (Oberley, L??au) or (waydert, C?/au) or (Smith, B?/au)
'?' TRUNCATION SYMBOL NOT VALID WITHIN 'L??AU'
'?' TRUNCATION SYMBOL NOT VALID WITHIN 'L??AU'
'?' TRUNCATION SYMBOL NOT VALID WITHIN 'L??AU'
'?' TRUNCATION SYMBOL NOT VALID WITHIN 'L??AU'
The truncation symbol ?  may be used only at the end of a search
term.  To specify a variable character within a word use '!', e.g.,
'wom!n' to search for both 'woman' and 'women'.  Enter "HELP
TRUNCATION" at an arrow prompt (=>) for more information.
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=> s (Oberley, L?/au) or (waydert, C?/au) or (Smith, B?/au)
L8       20146 (OBERLEY, L?/AU) OR (WAYDERT, C?/AU) OR (SMITH, B?/AU)

=> s l8 and (manganese superoxide dismutase or mnsod or catalase or cat or adcat or
phospholipid glutathione peroxidase or sod?)
L9         919 L8 AND (MANGANESE SUPEROXIDE DISMUTASE OR MNSOD OR CATALASE OR
               CAT OR ADCAT OR PHOSPHOLIPID GLUTATHIONE PEROXIDASE OR SOD?)

=> s l9 and antisense
L10         21 L9 AND ANTISENSE

=> dup rem l10
PROCESSING COMPLETED FOR L10
L11           7 DUP REM L10 (14 DUPLICATES REMOVED)

=> d l7 ibib abs 1-2; d l11 1-7 ibib abs

L7   ANSWER 1 OF 2  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
ACCESSION NUMBER:   1998:429492  BIOSIS
DOCUMENT NUMBER:    PREV199800429492
TITLE:              Na+/Ca2+ exchange in neonatal rat heart cells:
                    Antisense inhibition and protein
                    half-life.
AUTHOR(S):          Slodzinski, Martin K.; Blaustein, Mordecai P. (1)
CORPORATE SOURCE:   (1) Dep. Physiol., Univ. Md. Sch. Med., 655 W. Baltimore
                    St., Baltimore, MD 21201 USA
SOURCE:             American Journal of Physiology, (Aug., 1998) Vol. 275, No.
                    2 PART 1, pp. C459-C467.
                    ISSN: 0002-9513.
DOCUMENT TYPE:      Article
LANGUAGE:           English
AB   Cardiac Na+/Ca2+ exchanger (NCX) protein half-life (t1/2) and antisense
     knockdown were studied in primary cultured neonatal rat cardiomyocytes.
     Protein t1/2 was determined using (35S)methionine with a pulse-chase
     protocol. The 35S signal in NCX was identified by immunoprecipitation and
     Western blotting. The t1/2 of NCX protein was 33 h. Low concentrations
     (0.5 muM) of chimeric, phosphorothioated antisense oligodeoxynucleotides
     (AS-oligos) targeted to the region around the start
     codon of NCX1 transcript were used to knock down NCX protein and
     activity. Control myocytes (no oligos or scrambled oligos for at least 4
     days) exhibited spontaneous Ca2+ transients (measured with fura 2). The
     sustained ("diastolic") Ca2+ concentration in the cytosol ((Ca2+)cyt) of
     control cells was unaffected by cyclopiazonic acid (CPA) plus caffeine
     (Caf), which promote depletion of sarcoplasmic reticular Ca2+ stores, but
     (Ca2+)cyt rose in control cells when external Na+ was removed. In
     contrast, apprx60% of cells treated with AS-oligos for at least 4 days did
     not exhibit spontaneous Ca2+ transients or respond to Na+-free medium;
     however, CPA + Caf did induce a prolonged elevation in (Ca2+)cyt in these
     cells. In all cells, 50 mM K+ increased (Ca2+)cyt NCX protein was reduced
     by -50% in cells treated with AS-oligos for 7 days but was not reduced
     after only 2 days. These biochemical data are consistent with the
     physiological evidence of NCX knockdown in apprx60% of cells.

L7   ANSWER 2 OF 2  SCISEARCH  COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER:    1998:605903  SCISEARCH
THE GENUINE ARTICLE: 106WQ
TITLE:               Na+/Ca2+ exchange in neonatal rat heart cells:
                     antisense inhibition and protein
                     half-life
AUTHOR:              Slodzinski M K; Blaustein M P (Reprint)
CORPORATE SOURCE:    UNIV MARYLAND, SCH MED, DEPT PHYSIOL, 655 W BALTIMORE ST,
                     BALTIMORE, MD 21201 (Reprint); UNIV MARYLAND, SCH MED,
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DEPT PHYSIOL, BALTIMORE, MD 21201; UNIV MARYLAND, SCH MED, DEPT MED, BALTIMORE, MD 21201; UNIV MARYLAND, SCH MED, CTR VASC BIOL & HYPERTENS, BALTIMORE, MD 21201
COUNTRY OF AUTHOR: USA
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-CELL PHYSIOLOGY, (AUG 1998) Vol. 44, No. 2, pp. C459-C467.
Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
ISSN: 0363-6143.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 40

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Cardiac Na+/Ca2+ exchanger (NCX) protein half-life (t(1/2)) and antisense knockdown were studied in primary cultured neonatal rat cardiomyocytes. Protein t(1/2) was determined using [S-35]methionine with a pulse-chase protocol. The S-35 Signal in NCX was identified by immunoprecipitation and Western blotting. The t(1/2) of NCX protein was 33 h. Low concentrations (0.5 mu M) of chimeric, phosphorothioated antisense oligodeoxynucleotides (AS-oligos) targeted to the region around the **start codon** of NCX1 transcript were used to knock down NCX protein and activity. Control myocytes (no oligos or scrambled oligos for at least 4 days) exhibited spontaneous Ca2+ transients (measured with fura 2). The sustained (''diastolic'') Ca2+ concentration in the cytosol ([Ca2+](cyt)) of control cells was unaffected by cyclopiazonic acid (CPA) plus caffeine (Caf), which promote depletion of sarcoplasmic reticular Ca2+ stores, but [Ca2+](cyt) rose in control cells when external Na+ was removed. In contrast, similar to 60% of cells treated with AS-oligos for at least 4 days did not exhibit spontaneous Ca2+ transients or respond to Na+-free medium; however, CPA + Caf did induce a prolonged elevation in [Ca2+](cyt) in these cells. In all cells, 50 mM K+ increased [Ca2+](cyt). NCX protein was reduced by similar to 50% in cells treated with AS-oligos for 7 days but was not reduced after only 2 days. These biochemical data are consistent with the physiological evidence of NCX knockdown in similar to 60% of cells.

L11 ANSWER 1 OF 7 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2001:939497 SCISEARCH
THE GENUINE ARTICLE: 491RE
TITLE: Human **manganese superoxide dismutase** is specifically inhibited by **antisense** oligonucleotide **MnSOD** in human breast cancer cells.
AUTHOR: Weydert C J (Reprint); **Smith B B; Oberley L W**
CORPORATE SOURCE: Univ Iowa, Iowa City, IA 52242 USA
COUNTRY OF AUTHOR: USA
SOURCE: CLINICAL CANCER RESEARCH, (NOV 2001) Vol. 7, No. 11, Supp. [S], pp. 3681S-3681S. MA 137.
Publisher: AMER ASSOC CANCER RESEARCH, PO BOX 11806, BIRMINGHAM, AL 35202 USA.
ISSN: 1078-0432.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0

L11 ANSWER 2 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
ACCESSION NUMBER: 2001:138582 BIOSIS
DOCUMENT NUMBER: PREV200100138582

TITLE: Genes regulated in human breast cancer cells overexpressing manganese-containing superoxide dismutase.

AUTHOR(S): Li, Zhongkui; Khaletskiy, Alexander; Wang, Jianyi; Wong, Jeffrey Y. C.; **Oberley, Larry W.**; Li, Jian-Jian (1)

CORPORATE SOURCE: (1) Department of Radiation Research, Beckman Research Institute, City of Hope National Medical Center, 1500 Duarte Road, H115 Halper South Building, Duarte, CA, 91010-3000: jjli@coh.org USA

SOURCE: Free Radical Biology & Medicine, (February 1, 2001) Vol. 30, No. 3, pp. 260-267. print.
ISSN: 0891-5849.

DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The mitochondrial antioxidant enzyme manganese-containing superoxide dismutase (**MnSOD**) functions as a tumor suppressor gene. Reconstitution of **MnSOD** expression in several human cancer cell lines leads to reversion of malignancy and induces a resistant phenotype to the cytotoxic effects of TNF and hyperthermia. The signaling pathways that underlie these phenotypic changes in **MnSOD**-overexpressing cells are unknown, although alterations in the activity of several redox-sensitive transcription factors, including AP-1 and NF-kappaB, have been observed. To determine the downstream signaling molecules involved in **MnSOD**-induced cell resistant phenotype, in the present study we analyzed the expression profile of several groups of genes related to stress response, DNA repair, and apoptosis, in a human breast cancer MCF-7 cell line overexpressing **MnSOD** (MCF+**SOD**). Of 588 genes examined, 5 (0.85%) were up-regulated (2-42-fold), and 11 (1.9%) were down-regulated (2-33-fold) in the MCF+**SOD** cells compared to the parental MCF-7 cells. The five up-regulated genes were MET, GADD153, CD9, alpha-catenin and plakoglobin. The genes with the most significant down-regulation included: vascular endothelial growth factor receptor 1, TNF-alpha converting enzyme, and interleukin-1beta. GADD153 (involved in the repair of DNA double strand breaks) showed a 33-fold increase in microarray analysis and these results were confirmed by RT-PCR. To further determine the specificity in **MnSOD**-induced gene regulation, MCF+**SOD** cells were stably transfected with an **antisense MnSOD** sequence whose expression was controlled by a tetracycline-inducible regulator. Expression of three up-regulated genes was measured after induction of **antisense MnSOD** expression. Interestingly, expression level of GADD153 but not MET or CD9 was reduced 24 h after **antisense MnSOD** induction. Together, these results suggest that reconstitution of **MnSOD** in tumor cells can specifically modulate the expression of down-stream effector genes. GADD153 and other elements observed in the MCF+**SOD** cells may play a key role in signaling the **MnSOD**-induced cell phenotypic change.

L11 ANSWER 3 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:37530 BIOSIS
DOCUMENT NUMBER: PREV200100037530

TITLE: An **antisense** oligodeoxynucleotide to human **MnSOD** effectively blocks expression and enzymatic activity.

AUTHOR(S): Weydert, Christine J. (1); **Smith, Benjamin B. (1)** ; **Oberley, Larry W. (1)**

CORPORATE SOURCE: (1) Free Radical and Radiation Biology, University of Iowa, Iowa City, IA, 52242 USA

SOURCE: Free Radical Biology & Medicine, (2000) Vol. 29, No. Supplement 1, pp. S136. print.
Meeting Info.: 7th Annual Meeting of the Oxygen Society San Diego, CA, USA November 16-20, 2000

ISSN: 0891-5849.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L11 ANSWER 4 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2
ACCESSION NUMBER: 1998:183620 BIOSIS
DOCUMENT NUMBER: PREV199800183620
TITLE: **Manganese superoxide dismutase** protects nNOS neurons from NMDA and nitric oxide-mediated neurotoxicity.
AUTHOR(S): Gonzalez-Zulueta, Mirella; Ensz, Lisa M.; Mukhina, Galina; Lebovitz, Russell M.; Zwacka, Ral M.; F.engelhardt, John; **Oberley, Laary W.**; Dawson, Valina L.; Dawson, Ted M. (1)
CORPORATE SOURCE: (1) Dep. Neurol. Neurosci., Johns Hopkins Univ., Sch. Med., 650 N. Wolfe St., Pasthology 2-210, Baltimore, MD 21287 USA
SOURCE: Journal of Neuroscience, (March 15, 1998) Vol. 18, No. 6, pp. 2040-2055.
ISSN: 0270-6474.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Neuronal nitric oxide synthase (nNOS) neurons kill adjacent neurons through the action of NMDA-glutamate receptor activation, although they remain relatively resistant to the toxic effects of NMDA and NO. The molecular basis of the resistance of nNOS neurons to toxic insults is unknown. To begin to understand the molecular mechanisms of the resistance of nNOS neurons, we developed a pheochromacytoma-derived cell line (PC12) that is resistant to the toxic effects of NO. We found through serial analysis of gene expression (SAGE) that **manganese superoxide dismutase** (MNnSOD) is enriched in the NO-resistant PC12 cell-derived line (PC12-R). **Antisense MnSOD** renders PC12-R cells sensitive to NO toxicity and increases the sensitivity to NO in the parental, NO-sensitive PC12 line (PC12-S). Adenoviral transfer of **MnSOD** protects PC12-S cells against NO toxicity. We extended these studies to cortical cultures and showed that **MnSOD** is enriched in nNOS neurons and that **antisense MnSOD** renders nNOS neurons susceptible to NMDA neurotoxicity, although it has little effect on the overall susceptibility of cortical neurons to NMDA toxicity. Overexpression of **MnSOD** provides dramatic protection against -NMDA and NO toxicity in cortical cultures, but not against kainate or AMPA neurotoxicity. Furthermore, nNOS neurons from **MnSOD**-/- mice are markedly sensitive to NMDA toxicity. Adenoviral transfer of **MnSOD** to **MnSOD**-/- cultures restores resistance of nNOS neurons to NMDA toxicity. Thus, **MnSOD** is a major protective protein that appears to be essential for the resistance of nNOS neurons in cortical cultures to NMDA mediated neurotoxicity.

L11 ANSWER 5 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
ACCESSION NUMBER: 1995:547418 BIOSIS
DOCUMENT NUMBER: PREV199698561718
TITLE: The use of RT-PCR to distinguish between plasmid **MnSOD** transcripts and endogenous **MnSOD** mRNA.
AUTHOR(S): Li, Jian-Jian; Domann, Frederick; **Oberley, Larry W.** **(1)**
CORPORATE SOURCE: (1) Radiation Res. Lab., 14 Med. Lab., The Univ. Iowa, Iowa City, IA 52242 USA
SOURCE: Biochemical and Biophysical Research Communications, (1995) Vol. 216, No. 2, pp. 610-618.
ISSN: 0006-291X.
DOCUMENT TYPE: Article

LANGUAGE: English

AB We report here a convenient RT-PCR method to distinguish plasmid human **MnSOD** cDNA transcripts from the endogenous **MnSOD** gene products without engineering the cDNA insert. When a specific **antisense** primer for the carrier vector sequence was paired with a sense primer for the human **MnSOD** cDNA in RT-PCR analysis, a unique amplicon with the expected size was generated in **MnSOD** cDNA transfected cells but not in the wild type or vector control cells. The same primers were also used in genomic DNA-PCR to demonstrate genomic incorporation of cDNA in stably transfected cells. This method is convenient and specific in determining exogenous cDNA incorporation and expression in transfectants especially when transcripts of cDNA are difficult to separate from the endogenous mRNA by other methods.

L11 ANSWER 6 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 4
ACCESSION NUMBER: 1993:276279 BIOSIS
DOCUMENT NUMBER: PREV199396006504
TITLE: Increased **manganese superoxide dismutase** expression suppresses the malignant phenotype of human melanoma cells.
AUTHOR(S): Church, Susan L.; Grant, James W.; Ridnour, Lisa A.; **Oberley, Larry W.**; Swanson, Paul E.; Meltzer, Paul S.; Trent, Jeffrey M. (1)
CORPORATE SOURCE: (1) Dep. Radiation Oncol., Univ. Mich. Sch. Med., MSRBII C560 1150 West Medical Center Dr., Ann Arbor, MI 48109-0668 USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1993) Vol. 90, No. 7, pp. 3113-3117.
ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English

AB Introduction of a normal human chromosome 6 into human melanoma cell lines results in suppression of tumorigenicity. This suggests that a gene(s) on chromosome 6 controls the malignant phenotype of human melanoma. Because antioxidants can suppress the tumor-promotion phase of carcinogenesis, and because the antioxidant enzyme **manganese superoxide dismutase** (**MnSOD**) has been localized to a region of chromosome 6 frequently lost in melanomas, we have examined the effect of transfecting sense and **antisense** human **MnSOD** cDNAs into melanoma cell lines. Cell lines expressing abundant (+)-sense **MnSOD**-5 cDNAs significantly altered their phenotype in culture and lost their ability to form colonies in soft agar and tumors in nude mice. In contrast, the introduction of **antisense MnSOD** or +psv-2neo had no effect on melanoma tumorigenicity. These findings indicate that stable transfection of **MnSOD** cDNA into melanoma cell lines exerts a biological effect that mimics that observed after introduction of an entire human chromosome 6.

L11 ANSWER 7 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 5
ACCESSION NUMBER: 1989:514250 BIOSIS
DOCUMENT NUMBER: BA88:130393
TITLE: MANGANOUS SUPEROXIDE DISMUTASE IS ESSENTIAL FOR CELLULAR RESISTANCE TO CYTOTOXICITY OF TUMOR NECROSIS FACTOR.
AUTHOR(S): WONG G H W; ELWELL J H; **OBERLEY L W**; GOEDDEL D V
CORPORATE SOURCE: DEP. MOL. BIOL., GENENTECH, INC., 460 POINT SAN BRUNO, BOULEVARD, SOUTH SAN FRANCISCO, CALIF. 94080.
SOURCE: CELL, (1989) 58 (5), 923-932.
CODEN: CELLB5. ISSN: 0092-8674.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB Tumor necrosis factor (TNF) induces the synthesis of protein(s) that can protect cells against subsequent killing by TNF in the presence of

cycloheximide. Here we demonstrate that manganous superoxide dismutase (**MnSOD**), a mitochondrial enzyme involved in the scavenging of superoxide radicals (O2-), is such a protein. Overexpression of **MnSOD** confers increased resistance to TNF plus cycloheximide on the 293 human embryonic kidney cell line. Conversely, expression of **antisense MnSOD** RNA renders these cells sensitive to TNF even in the absence of cycloheximide. The TNF sensitivity of the ME-180 human cervical carcinoma cell line can also be modulated through expression of sense and **antisense MnSOD** RNAs. These data identify **MnSOD** as an important determinant of cellular resistance to TNF and implicate motochondrially generated O2- as a key component of TNF-mediated tumor cell killing.

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